Hair

Hair cycle: ⊙ Autonomous, rhythmic transformation of Fully developed hair follicle through phases of regression, growth, resting Controlled by the Follicle itself,

⊙ hair doesn't grow in the same time there is unsynchronization (some hair grow, some hair lost)

⊙ 4 stages are Known:

① Anagen phase → active stage
② Catagen phase → involuting stage → involution of Lower ⅓ of hair follicle by massive Keratinocyte apoptosis
③ Telogen phase → Resting phase then new hair regrow
④ Exogen phase Active hair shaft shedding (anagen IV)

Total number of Humman hair follicle is 5,000,000 (Mostly vellus follicle)

100,000 in scalp ← 85% anagen, 0.5–1% catagen, 15% telogen

Duration of hair cycle For example scalp ← Anagen 3 years (2–6), Catagen 3 weeks (2–3), telogen 3 month

NB → Estrogen prolong anagen.
→ Thyroxine promot growth
→ Corticosteroid retard anagen onset

Hair growth: 0.35 mm / day i.e 1cm / month

Ⓐ Hair production is not increased by Cutting / or shaving
Ⓔ Estrogen ↓ hair growth rate
Ⓐ Androgen ↑ " " " , hair diameter

hair growth is mediated by testosterone.

testosterone —5α-reductase→ DHT → promot Conversion of Terminal hair to vellous hair → hair loss

Finasteride, dutasteride are 5α reductase inhibitors.

Hypertrichosis

- Increase the hair growth of body hair (Non-androgen dependent) with normal androgen metabolism
  - ① Generalized
  - ② Localized

[1] Generalized Hypertrichosis

ⓐ Hypotrichosis Lanuginosa

Congenital

AR, Fetal hair not replaced by vellus hair but → persist grow and may cover whole body

Acquired

Fine lanugo hair grow over large area of the body replacing normal hair associated ε' paraneoplastic S ε malignancy

ⓑ Pigmented/terminal hair hypertrichosis

Congenital generalized

- Generalized hypertrichosis + Gingival hyperplasia
- Skeletal defect,
- mental retardation.
- other congenital anomalies.
- Maternal ingestion of minoxidil.

prepubertal

- offect face (forehead pre auricular area)
- proximal extremities and back
- hair in the back in (inverted fire tree) pattern.

Drug-induced

- Minoxidil
- D-penicillamine
- PUVA
- streptomycin

[2] Localized Hypertrichosis

① Congenital localized:
- Hamar tomas (Congenital melanocytic nevi_ Becker's nevi
- Nevoid hypertrichosis
- Faun tail: hypertrichosis in Sacral area a sign of spina bifida.

② Hereditary hypertrichosis:
affecting the specific anatomic sites:
- Hypertrichosis Cubiti (Hairy elbow syndrome)
- hairy palm, sole, auricle, eye brow.

③ Localized Hypertrichosis :-
in hereditary and acquired diseases eg. Hypertrichosis with sun exposed areas. is a sign of Prophyria. (PCT)

④ Acquired circumscribed Hypertrichosis :-
after trauma eg surgical wound
after inflammation due to prolonged CST use.

D.D of Hypertrichosis : is Hirsutism

Lab invest : Normal androgen Level.

Treatment of Hypertrichosis :

① Cosmetics :- hair bleaching ∂ 6% H_2O_2
- Shaving
- Electrolysis, thermolysis
- Laser hair removal
- Intense pulsed light (IPL)

② stop using of drugs (minoxidil)

③ Rx of underlying cause (malignancy)

④ Anti-androgens if associated ∂ elevated androgen level.

Hirsutism

excessive growth of coarse, terminal hair in woman on androgen dependent areas of the body

Classification

[1] Constitutional
[2] Endocrine - organ based Hirsutism
[3] Iatrogenic
[4] Ectopic hormone production
[5] hepatic Hirsutism
[6] Failure of Converting Androgen → Estrogen

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(1) Constitutional
- Familial → Normal Hormon Level (end organs is more sensitive to androgens)
- Adrenal → Mild ↑ & DHEA-S (Di hydroepi andro sterone sulfate)
- Ovarian → Normal DHEA-S + ↑ Free testosterone
- Hyperprolactemia → ↑ prolactin H.

(2) Endocrine - organ (H)
- Adrenal → thin female, Central hirsutism
  - (a) Non-tumoural adrenal hirsutism → Adrenal hyperplasia → Hyper cortisolism Cushing ↓
  - (b) Tumour adrenal hirsutism, Adenoma, Carcinoma.
- ovarian → obese woman + Menstrual disturbance; Lateral Hirsutism
  - (a) Non-tumaural PCO.
  - (b) tumour ovarian tumours
- pituitary → ↑ ACTH ↑ prolactin

(3) Ectopic Hormone production
- Lung Carcinoma → ACTH secretion
- chorio carcinoma → BhCG secretion.

(4) Iatrogenic Hirsutism → Steroid adminstration

(5) Hepatic Hirsutism Liver disease → ↓ sex hormone binding globulin → ↑ testosteron Free → ↑ DHT

(6) peripheral Failure of Conversion of Androgen to estrogen lead to ↑ Free androstenedions.

Management of Hirsutism

① history: age of onset, Family history, menstruation, medical history

② Examinations - ⊙ Degree of hirsutism ~~Ferr~~ Ferriman, Gallwey score from 1–4 ① No hair in chest ④ hair in chest, abdomen, back, thigh.

⊙ other signs of virilism → Acne, Frontal balding, ↑ muscle mass

⊙ CT, MRI, U/S of pelvis, abdomen to detect tumours.

[3] Lab investigation:

- total Testosterone > 200 ~~ng~~/ml → Androgenic tumour
- DHEA-S > 700 ng/dL → adrenal tumour
- ↑ 17-hydroxy progesteron → Congenital Adrenal Hyperplasia.
- PCO ↑ testosterone, ↑ LH, LH:FSH > 3:1
- ACTH stimulation test: 250 µg IV infusion ⁂ if > 1000 ng → +ve test
- Dexamethasone suppression test.

[4] Treatment ① Cosmetic: bleaching, shaving, laser, IPL

② R/ of neoplastic causes: surgery – irradiation – chemotherapy

③ R/ of Non Neoplastic causes:

⊙ Suppression of adrenal androgens:
- Dexamethasone 0.25–0.5 mg/d for 3 months.
* Prednisolone 7–5 mg/dg/2 months then 5 mg/d/2 months then 2 mg/day / 6 months.

⊛ Suppression of ovarian androgens:
* oral Contraceptive pils
* ~~GnRH~~ GTRH antagonist / 6 months

⊛ Androgen receptor blockers:
* Spironolactone 50–200 mg/day / 6 months
* ~~Cyp~~ Cyproterone acetate (Androcur) 20mg–100 mg form day (5) to day (14) of the cycle.

⊛ * 5α reductase inhibitor → Finasteride 5mg/day

⊛ * Ketoconazol 400–1200 mg/day.

⊛ Suppression of pituitary prolactin production (Cabergoline – Bromocriptine dostenix

⊛ Metformin in PCO

⊛ Topical therapy eflornithine

* Surgical for ovarian, adrenal Tumours.
* Stop iatrogenic causes.

Alopecia

loss of hair from hairy regions of the body.

Evaluation of Alopecia

① history ② Examination ③ Investigations.

① History: shedding or thinning

① * shedding hair falling is sever daily (TE, AnagenE,
* thinning of hair lead to visible scalp. but without noticeble hair falling. eg. AGA or Senile Alopecia

② Duration: Congenital or acquired

③ Family history: hair shaft disorders, AGA

④ hair care procedures: dyes, bleaching, cosmatics .....

② Examination: Patterned circumscribed (Patch) eg A.A

① hair pattern of hair loss → diffuse: T.E

② hair fragility due to hair shaft disorder: Trichorrhexis nodosa

③ Scalp surface. to see inflammation lead to alopecia
eg: psoriasis, histiocytosis.

④ Examination of other hairy region.

③ Investigations:

① Gentle hair pull test: 50-60 hair grasped by fore finger and thumb then pull without causing pain
Normal < 10% of hair pulled are extracted
5 hairs or less. if more than 10% → T.E.
the female shoudn't wash her hair 24-48 h befor test

② Forcible hair pluck test (Tricho gram) (hair root status)
bundle of 50-60 hair is pulled out by artery forceps and the extracted hair is examined under microscope
the female should be advised not to wash her hair 5-7 days befor the test.

Anagen hair: the root is the largest at it's base
the inner root sheath is present and Firm

telogen hair: Club shaped rood, lack of angulation, loose sheath.

Dystrophic hair: pathological conditions. thin, without root sheath, tapper at proximal end.

Normal trichogram:
the scalp contain 100,000 hair follicle.
85% in anagen phase
0.5–1% in Catagen phase
15% in telogen phase.

Normally 25–100 hair (telogen) lost per day.

(c) Tricho Scan: Automated software program responsible fa analysis of hair growth.

(d) Time shed_hair Caunt:
- Colleting hair loosed in 24 h (Normally 50–100)
- brush hair for 1 minute. Count the number of hair Shed hair Normal(50) if >150 active hair loss.

(e) Hair growth window:
For pt who say that their hair doesn't grow.
Shave area 2x2 cm after one week it should be
7 X 0.35mm = 2.5 mm.

(F) Scalp biopsy: to examine the presence of inflammation or scarring. two 4mm punch biopsies are examined.

(g) Dermo scope

(H) lab investigation: CBC, s. iron, ...
for infection: Syphilis, KOH → Fungal

Cicatricial Alopecia

Occurs as a result of destruction of hair follicle by scar tissue leading to irreversible alopecia

1ry: the hair follicle is the target of inflammation

2ry: Follicles are destroyed in a non specific way (burn - trauma)

Causes of cicatricial Alopecia:-

① Congenital: Ichthyosis (Recessive X-Linked)
Darier's disease - epidermal nevi
epidermolysis bullosa (Recessive dystrophic)

② Acquired:

ⓐ physical injury: Mechanical trauma, burn, Radiation

ⓑ infection → Fungal Kerion, Favus viral: Varicella, HZ
bact.: LV, gumma, Leprosy, Carbuncle, acne necrotica
protozoa: leishmaniasis

ⓒ Tumours BCC, SCC, metastatic Carcinoma.

ⓓ CTCL

ⓔ Collagen disease DLE, Morphea, DM

Ⓕ Dermatosis of unknown eitology: LP - Sarcoidosis
Cicatricial pemphigoid - Acne Keloidalis
pseudofolliculitis barbe - Pyoderma gangrenosum.

⑨ Clinical syndromes: pseudopelade of Brocq
folliculitis decalvans.

Pseudopelade of Brocq

Woman to man 3:1

Condition in which there is chronic patchs of alopecia which are slowly progressive, irregular defined, affecting the crown or back of the head. early stage: patches with mild perifollicular erythema. late stage: patch smooth shinny atrophy without sign of inflammation and absence of follicular pores. DIF -ve or minimal IgM at BMZ of infundibulum

H/P moderate perifollicular infiltrate (lymphocytic) around upper ½ of follicle
Later: the epidermis (thin, atrophic) dermis is (Sclerotic)
Collagen bundles run vertically to the skin mark Follicle/erector pilli in place

Classification of Iry cicatricial Alopecia.

	Scalp	Treatment
Lymphocytic		
① DLE	Symptomatic, erythematous scalp plaques with follicular plugs. telangeiectasia, atrophy depigmentation & time activity in the center of alopecia patch	@IL Corticosteroid + Topical CST ⊙ Hydroquinone prednisolone, tacrolimus - tazarotene isotretinoin.
② Lichen planopilaris	pruritic, multifocal or central alopecia patches & follicular hyperkeratosis, erythema at hair bearing margin	⇓ ⊕ cyclosporine griseofulvin Anti androgens (Finasteride) and dutasteride)
ⓐ classic		
ⓑ Frontal Fibrosing Alopecia	Fronto temporal recession of hair at hair bearing margin, eye lash loss, Fascial papules, and body hair involvemet in severe form.	
③ pseudopelade of Borcq	Asymptomatic, non inflammed ivory-white or flesh Coloured small oval round confetti-like reticular or large irregular patches ± Atrophy	⊕ Topical CST I L T A ⊙ predni solone ⊙ Hyydroquinine. Isotretinoin.
Neutrophilic		
@ Folliculitis Decalvans	none in central scalp, grouped follicular pustules Military abscess or hair bearing margin	Abcs + steroids. - Rifampicin + 2nd Abcs - Fucidic acid + Zinc
Mixed		
Acne Keloidalis	Occipital scalp (nape) firm red-brown papules, papulopustules, Nodules, Keloidal plagues	@IL TAc ± Antibiotics ⊙ Excision of Plaque form.

Telogen effluvium

A condition of diffuse hair loss in which the anagen hair follicles enter into telogen phase prematurely leading to diffuse shedding. usually occures after sever stress.

Causes of TE:

① Physiologic
- Physiologic effluvium of New born
- Post partum effluvium.

② Stress or injury:
- High fever (Malaria)
- Sever infection (HIV)(syphilis)
- chronic diseases: Liver, Renal failure, SLE,
- Hypothyroidism.
- Crash or liquid protein diet.
- Mal nutrition
- iron def. anaemia
- zinc def.
- Major surgery.

③ Drugs: Retinoids – Heavy metals – β-blockers – hormmons – Antithyroid – Anticonvulsant – Anticoggulant (heparin)

Acute TE: diffuse shedding of hair occurs 3-4 months after stress exposure. it continue for few months and regrowth take place within ⑥ months. good prognosis

Chronic TE: Telogen hair shedding is longer than ⑥ months.

pathogenesis

[1] Immediate Anagen Release] Follicles enter telogen phase prematurely → Acute Telogen effluvium

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[2] Delay anagen Release — in pregnancy hair follicle remain in anagen phase and post partum it → convert to telogen result in ↑ shedding → TE → Post partum hair loss

[3] Short anagen syndrome	Shortening of anagen phase so club hair are released 4-6 week after the onset of anagen	AGA and Chronic idiopathic TE
[4] Immediate telogen Release	Shortening of Normal telogen	due to drugs eg Minoxidil
[5] Delay telogen Release	prolonged telogen followed by transition to Anagen	In some Humans

Lab ~~Investigation~~ : Diagnosis

History - Examination - investigation

[1] History :- Duration of shedding.
- History of Surgey, Fever, Stress, Pregnancy.
- History of Chronic illness Liver, Renal, malignancy....
- Menstrual history - Diet history.
- hair care procedures.

[2] Examination :- inspection (Manual - Wood's light, dermoscope).
- Inflammation → ery thema, Scaling, infection
- Hair collection Hair pull, hair pluck, Hair clipping
Nail examination.

[3] Lab investigation

CBC	CMP	S. iron	
TSH	T4	S. zinc	vit. D

Treatment of the cause, the underlying disease + Nutritional supplement + Minoxidil 2% or 5%

Androgenic Alopecia

Progressive transformation of terminal Follicles into vellous Follicles. it's induced by androgen stimulation of genitically predisposed persons.

pathogenesis: ① Genetic AD. the inheritance is polygenic female has less family history.

② Androgens: there's relation bet AGA and androgens castrated male before puberty don't develop baldness but they develope baldness if they receive testosteron

③ Aging: baldness is progressive by aging.

Diagnosis:

① Family history (Maternal, Paternal)

② Trichogram and scalp biopsy: ⊙ Normal total number of Follicles with ↑ propotion of telogen hair

⊙ Reduction of the size of hair Follicle → ↓ diameter

Terminal hair: vellous hair reduce >8:1 to <4:1

Anagen : telogen ratio reduce from 12:1 to 5:1

③ Lab test: for androgen ↑ in female if manifestation of hyperandrogenism is present.

Clinical Features:

Male: start with bitemporal recession followed by balding of vertex, sparing the post, lateral scalp margins

Female: Start in late life, not sever as males. it's usuually diffuse than patterned (thining) affecting frontovertical area but unlike men - the frontal hair line retains. widening of central part shows christmas tree pattern

Grading according central part widneing (Ludwig)

Grade I minimal widening Grade II moderate Grade III significant widening + thining

Treatment :

① Systemic Antiandrogens

② Finastride
1% for males FDA approved
2-5% For females Not FDA approved

③ Topical
Minoxidil: 2% FDA approved For Female and male
5% " " " Male only.
Combination with tretinoin enhance the trichogenic effect of minoxidil
Vasodilater
↑ Cutaneous Blood Flow to sclop.
↑ Conversion of vellus Hair to terminal hair in 30% of pt's

④ light therapy: ↑ vascular circulation, ATP production
Enhance matrix cell proliferation.
665 nm/red light (HairMax - laser Comb)

⑤ Cosmotic Rx hair transplantation.

⑥ Prostaglandin analogues:
Topical Latanoprost 0.1% ↑ Hair density
Injection of bimatoprost 0.03% weekly for 12 week
good for Fenal pattern Hair Loss
FPHL

Alopecia Areata

Common skin disorder, non scarring alopecia present as circular area of alopecia which can lead to total scalp hair loss → Alopecia totalis or Complete scalp and body hair loss → Alopecia universalis

Etiology:

① Genetic: ⊙ 20% have +ve family history.
- ⊙ Associated ć Congenital disease (Down's Syndrome)
- ⊙ Associated ó Atopy
- ⊙ Associated ć HLA D@7, D@3, DR11

② Immunological: ⊙ AA is an autoimmune disease
auto-Antigen is most propably melanocyte associated Antigen
- ⊙ Associated ć other autoimmune disease: LP, Vitiligo

③ Emotional stress.

④ Hormonal fluctuation, infection, vaccines are triggering factors of A.A.

Histopathology:

① presence of miniature hair structure of early anagen or telogen

② peribulbar lymphocytic infiltrate (Swarm of bees) mainly T-Helper type. CD_4/CD_8 is higher indicate activity

2 stages:

ⓐ Early stage (Progressive): MN cell infiltrate around bulb + hair matrix changes + trichomalacia + narrowing of hair shaft.

ⓑ Stable disease: Scanty peribulbar infiltrate arrested anagen hair.

Clinical Features:

Sudden and complete Hair loss in a circumscribed area in which the skin is completely Normal.

Exclamation mark hair: found around the patch which indicates progression.

this hair shaft is thin proximally and thick distally that can be easly pulled-out ⇒ !

ophiasis: Extension of A.A along the scalp margin (active)

Alopecia totalis: total scalp Hair loss

Alopecia universalis: total scalp hair and body Hair loss.

AA diffusa: AA associated & diffuse hair loss as TE

Reticular AA:- Recurrent patchy disease.

Sites: Scalp - beard moustach eye brow.

Trichogram: Normal or telogen Hair root but if progressive rapidly ⇒ telogen dystrophic

Associations ĪVĀG

① Atopy ② eye changes → Cataract & Alopecia totalis

③ Nail changes: 20% of cases

pitting + longitudinal ridging + thickening the intensity which parallel of hair loss.

Course and prognosis is variable

bad prognosis in ĪVĀG

1. Atopy
2. Ophiasis
3. Multiple lesion
4. Presence of exclamation mark.
5. affection of eye brow
6. Nail changes.

Treatment of A.A :-

① Topical
1- Topical or Intra Cataneous CST Kenacort
2- Non specific irritant Tincture iodine, Tr capsicum
3- Photo chemotherapy PUVA
④ ~~Monixidil~~ Minoxidil
⑤ Anthralin
⑥ RPP
⑦ Prostaglandine analogues Latanoprost, bimatoprost

② Systemic therapy :
① systemic CST only in rapid progressive active A.A
prednisolone 0.5-0.8 mg/kg/dg slowly traped over 2 months
② systemic cyclosporine
③ Methotrexate
④ Biologics (infliximab - etanercept) (failed)

Drug induced alopecia

diffuse- non scarring - reversible alopacia. in scalp

mechanism:

Direct effect on hair Follicle

interruption of anagen growth (anagen effluvium)
Chemotherapy
Anti Cancerous
• Colchicine

premature Pericephitation of Telogen (T-E)

Androgenic induction of normal terminal into vellous hair
(Androgenic Progesterone, Anabolic steroid, exogenous androgen

Indirect
drug induce systemic disorder → Hair Loss
Hypothyroidism
Lichenoid drug eruption
↓
Cicatricial alopecia or TEN

Trichotillomania

Psychological disorder. the pt has habit of twisting the hair around his finger and pull it out.

females > males 5:1 child > adult 7:1 boys > girls.

C/P ill-defined area (patch) in which the hair is twisted and broken of various distance of normal skin scalp.

Sites: eye brow – eye lash – pubic hair.

pathogenesis: ~~according to the~~ Impulse control disorder, causing sever stress or impairment.

Criteria of Trichotillomania

1. recurrent self hair pulling out result in hair loss
2. increase tension immediately befor pulling out the hair
3. pleasure or relief sensation after pulling out the hair

Trichogram in affected area: ↓ telogen hair + 2ry trichodystrophies with fracture ~~end~~ distal end. Un affected area is Normal

Growth window: Steady increase in hair density. in the shaved area.

H/P * Distorted follicular anatomy without inflammation.
* Multiple Catagen follicles * pigment casts and Keratin plugs (Trichomalacia)

D.D of

1. Scaly ringworm: Trichotillomania (No scaling + -ve fungal culture)
2. Alopecia areata: in Trichotillomania there's NO exclamation mark.

R/ Psychatric – (minor or Major tranquilizer (SSRI)